(liquor aluminum acetate, diluted 1-8; 1-15) are of value. A soothing lotion, such as calamine, is frequently all that is necessary. A paste, such as Lassar's, with a quarter to a half per cent of phenol for its antipruritic effect, is of value.

ATOPIC DERMATITIS

This is the classical form of infantile eczema. The face and flexor surfaces of the extremities are the sites of predilection. It is characterized by weeping, crusting, lichenification, and extreme pruritus. It is in this form that the diet is of particular importance, as will be brought out by others in this symposium. External irritants, such as feathers and wool, also play an important rôle, as in the contact type. For the weeping and crusting stage, the method of choice is compressing or applying massive wet dressings. Treating large areas is best done in the hospital. A few layers of moist gauze or cotton is not a wet dressing, because the exudate will stick to it and irritate the area and when it dries it does more harm than good. The dressings should be made from unstarched gauze or soft linen, saturated with the solution to be used and wrung out so that it is not dripping. This is placed on the affected areas, and should be at least two inches thick. The dressing is then covered with some sort of impermeable material, such as cellophane, to keep the dressings from drying. A dry bandage or towel is then put on to hold the dressing in place. To put such a dressing on the face requires considerable nursing skill. When improperly applied, a wet dressing is worse than none. Dressings should be changed every three to four hours. A wet dressing can be made with saturated boric acid solution, Burow's solution 1-8, 1-20; potassium permanganate 1-5000 is an excellent solution if any secondary infection is present. After weeping and crusting have subsided, which should be in a few days, ointments are in order.

By far the most useful single remedy is crude coal-tar. One should make sure that eastern crude coal-tar is being used, and details should be given to the pharmacist if he does not know how to make the preparation. It should be smooth and dry. If it is not properly prepared, it crumbles and tends to aggravate the skin. Its proper preparation is with equal parts of crude coal-tar and zinc oxid mixed together. Then starch and petrolatum are mixed. These two mixtures are then worked in together to form a smooth black paste. This is applied gently to the affected areas, if there is any weeping and erythema present. If the areas are lichenified, the ointment should be rubbed in.

Prescription III. Crude coal-tar ointment

Crude coal-tar	4.0 grams
Zinc oxid	4.0 grams
Mix and add	
Starch	30.0 grams
Petrolatum	30.0 grams

Mix and label: Apply to affected areas two or three times a day.

Crude coal-tar can be used in full strength, the liquid being painted on the affected areas every other day. After the affected areas begin to show slight exfoliation, the tar should be stopped, and a simple paste of equal parts of boric acid ointment and Lassar's paste should be used. If the tar is used too long a dermatitis may develop. This is frequently of a follicular type. Another preparation which has been useful in my hands is a 10 per cent ichthyol incorporated in Lassar's paste. This is less irritating than crude coal tar. Naftalan, a mild tar used in 5 to 10 per cent strength, has the same indications as ichthyol.

COMMENT

No attempt has been made to include all the various remedies that are of value in the different types of infantile eczema. One treating these cases should become completely familiar with the medications he is using, so that the changing of only one or two per cent of any one ingredient in the course of the disease will mean involution, and not evolution, of the process. There are a few general statements that may be made:

1. Change of environment (hospitalization) is beneficial.
2. Removal of wool and feathers as common cause of contact reactions should always be done.
3. Dermatitis from local medication should be watched for. Mercury is the worst offender.
4. Weeping areas should be treated with wet dressings or compresses before lotions and ointments are used.
5. On inflamed areas, pastes are better than greasy ointments.
6. Recurrences are frequent, some unexplainable and others due to carelessness in following detailed directions.
7. Always remember that great majority of infants clear around the age of two years.

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EPILEPSY*

A GENERAL SURVEY OF THE CONVULSIVE STATE

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CONVULSIONS occur in a truly bewildering variety of conditions. Paresis, brain tumor, diabetes, alcoholism, brain trauma, hysteria, uremia, and tetanus are but a few of fifty or more organic, toxic and functional disorders associated with convulsive manifestations.

Because of the mystery surroundinng its etiology, the title of idiopathic epilepsy was given to a group of so-called spontaneous seizures; but just as fever and pain are symptoms of an underlying pathology, so should the epilepsies, or convulsive states, be considered.

The finding of neolithic skulls, with trephine openings to allow of the escape of the devils caus-

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ing epilepsy, antedating the written records of man, speaks for the antiquity of the disease. In the United States today, with an estimated 600,000 persons suffering from convulsive disorders and with over 40,000 of these confined in state psychiatric institutions,[1] it still remains one of the great social and economic problems.

In the state hospitals of California last year there were 22,608 patients.[2] Of these, 665 were epileptics whose yearly cost has been estimated at $151,620. During the year, one hundred new cases of epilepsy were admitted. Of these patients, none had received a collegiate education, only eighteen had gone through high school, and 48 per cent were entirely dependent. There were thirty-six deaths among the epileptics and of these twenty had been in the hospital for an average of twelve and one-half years. These figures give but a small idea of the seriousness of the situation.

Those suffering from convulsive disorders and yet not hospitalized still remain a handicapped group. As automobile operators they are a serious problem, and their choice of occupation is necessarily limited, inasmuch as their sudden seizures cause a hazard both to themselves and to others. In large colonies these people do quite well together. At Agnew a practice is made to have male epileptics work on the lawns in groups of twos and threes. If one falls during a seizure, he has a soft carpet on which to fall, and the other patients in the group care for him until he is again ready to carry on. In this way they may even take trips into the country.

DIAGNOSIS

The diagnosis of epilepsy in a state hospital case is usually not difficult. In its incipiency, however, this may be fraught with much uncertainty. Fainting spells, with convulsive movements, may resemble epilepsy very closely. Indeed sleep, faints, syncopy, convulsions, petit and grand mal epilepsy, all have a strikingly similar underlying mechanism.

To elaborate further: sleep, as pointed out by Rosett,[3] does not occur all at once but step by step through the sensory, associative, and motor fields. Thus, as one lies down to sleep the body is no longer in need of vestibular help to maintain equilibrium, visual stimuli cease, and auditory sensations are cut to a minimum. This allows the associative and thalmic fields to fill the mind with vivid and inaccurate memory images. As these fade, the motor area, at first active, next recedes, and the individual becomes, as it were, decerabrated. It is often possible at this stage to obtain a positive Babinski, and it is here that the familiar jerking movements are so apt to occur. Next the muscles become flaccid, no deep reflexes are obtained, the last neuronic pathway through the stem and cord is closed, and the individual passes into a profound stage of sleep. Awakening proceeds in the reverse order.

STAGES OF AN EPILEPTIC SEIZURE

The various stages of an epileptic seizure can, according to Rosett,[3] be closely correlated with those of sleep. Thus, a typical seizure is frequently ushered in by an aura with an accompanying narrowing of the sensory fields. One patient, E. S., described this aura to me as follows: "I feel funny, things seem to be floating before me, it's like looking through a fog. I seem to be reaching out for something, and then falling into space, dark space."

Following this suppression of the sensory field, associative and thalmic release next ensues, bringing with it vivid imagry and hallucinations, with an increased feeling tone. Patient G. V. explained the sensory aura to me as follows: "I can tell when it is coming; I feel ill in the pit of my stomach and a hot feeling comes in my left ear." Then follow hallucinations of hearing with a feeling of fear, as she puts it: "I get afraid and have a feeling that men are coming up from behind to grab me, and I can hear them say: 'Come on, we are going to get you.'" The patient next becomes unconscious as the wave passes on, and with the loss of the cerebrocerebellar pathway there ensues a state of tense muscular rigidity known as the tonic phase of the convulsion. Following this comes the jerking clonic convulsive movements of the whole body. At about the time these generalized convulsions are over, it is frequently possible to obtain a positive Babinski, indicating that the corticospinal pathway through the pyramidal tract has been extinguished. Following the convulsion, the muscles lose their tonicity and become flaccid, showing again that the last and most fundamental pathway of stem and cord has been blocked out. Recovery, as in sleep, proceeds in the reverse order: First, the muscles regain their tone, movements take place, dreams are common, and the patient, though at first confused, regains his usual conscious state once more.

This wave of disability may recede at any stage of the seizure and cause the syndrome to be only partially completed.[3] Thus one may have only the preliminary aura without further trouble, or it may go on to a momentary loss of consciousness without proceeding to the convulsive stage. Thus patient A. Z. came to my office one day and, in a somewhat euphoric mood, began telling me how long it had been since he had had a convulsion. "Doctor," he said, "it's been at least six months since I've had a spell, yes, sir, at least six months." Suddenly, with a far-away look in his eyes, he turned, walked to the corner of the room, picked up a feather duster and, holding it before him as he would a candle, made a tour of the room as if looking for some lost object. Then, replacing the duster in the corner he came back, and as he focused his gaze on me once more, continued: "Yes, doctor, it's been all of six months since I've had a spell." He had, of course, just experienced an epileptic equivalent of which he himself had no appreciation.

OTHER PHASES

As stated above, faints are often accompanied by convulsive movements, and are hard to distinguish from light attacks of epilepsy. Fainting is often preceded by the feeling that everything in the environment is floating away. Daylight seems to be a long way off and, finally, complete blackness ensues. Gowers cites several cases in which fainting attacks eventually developed into true epilepsy.[4]

It is interesting and significant to note that dizziness, syncopy, and fainting can be caused by pres-

sure on the carotid sinus. It is also suggestive that, by exerting more and more pressure on the sinus, convulsions can be produced. The symptoms caused by this pressure, according to Smith,[5] are aura with spots before the eyes, epigastric distress, light-headedness, unconsciousness, and mild convulsions—the typical syndrome of epilepsy. According to Weiss,[6] in some cases carotid pressure causes a slowing of the heart and a lowering of the blood pressure. In other cases it neither lowers the blood pressure nor slows the heart, but acts directly on some brain center, probably producing a sudden change in the cerebral vessels, consisting of a contraction followed by dilatation, thus accounting for the syndrome. The occurrence of dizzy spells, fainting spells, and generalized convulsions were found in 16 per cent of a series of one hundred consecutive cases of senile and arteriosclerotic psychosis, as reported by Tompkins.[7] Of these, 6 per cent were of the epileptoid character, 7 per cent were convulsive, and 3 per cent showed a profound and lasting change in consciousness. It would be hard to believe that these dizzy spells, faints, and convulsions were not all an integral part of the convulsive state.

Convulsions can likewise be produced by drugs, such as insulin and metrazol, which are used extensively in the shock therapy of schizophrenia. These convulsions resemble epileptic seizures and can, of course, precipitate attacks in those suffering from epilepsy.

COMMENT

Great progress has been achieved in brain surgery through the use of delicate electrodes in producing convulsions by spotting the aural center which sets off the convulsive explosions, but much argument has arisen as to what course these convulsion-producing waves pursue. Penfield[8] likens the condition to a cyclone, which sweeps through the brain, causing wild, uncoördinated muscular contractions.

Recently, however, Erickson[9] has demonstrated quite convincingly that, in monkeys, convulsion-producing waves follow along the motor convolution from one contiguous area to the next, probably by means of the short intracortical association fibers. When this wave—*e. g.*, one passing up the convolution from the arm to the leg area—is interrupted by a transverse ligature, it does not jump the gap and continue up to the foot center, but crosses by way of association fibers through the corpus callosum to the corresponding arm area of the opposite hemisphere. Here it continues a course up this convolution to the leg center, where it recrosses above the ligature and into the leg area of the original hemisphere.

This theory of the progression of waves would seem to apply to the sensory as well as the motor area if the description of an aura, as given by my patient B. B., is correct. She stated that she first noticed a numbness arising in the toes of the left foot. This numbness proceeded up the leg into the thigh, along a narrow band from the thigh to the left shoulder, thence down the arm and out into the fingertips of the left hand. (This patient was left-handed.) It will be noted that this description follows in sequence the contiguous sensory areas as mapped out by cerebral experimentation. Occasionally, this patient said, when the sensation reached the left hand there followed almost simultaneously a feeling of numbness in the right hand. Efforts had been made by teachers to make this left-handed individual right-handed. This suggests that a deepened pathway through the corpus callosum might account for the occasional crossing of the aural wave from the left to the right hand.

The fact that so many seizures occur at night would also suggest that, through sleep and the abeyance of the sensory field, the first step in the neuronic arc toward the convulsive explosion has already been taken. The powder seems to lie somewhere along the associative, thalmic, or motor pathway. It might even reside in some localized irritative lesion of the sensory area, where it remains ready to be set off by the constant pulling of this trigger.

Why these waves should start, no one as yet knows. Starr[10] cites the case of a young girl who was suddenly awakened at night by her brother, who had covered himself with a white sheet. She was much alarmed and one hour later had a major attack. Following this she became a confirmed epileptic. Fright was given as the cause in this case, yet the fact remains that ordinarily no one would develop epilepsy from such a fright. However, it might have been the precipitating factor in a subject potentially an epileptic. Such seems to have been the case in a diminutive-sized schoolgirl, M. R., whom I saw playing a trombone in an orchestra. At the end of a long number in which she had played a trombone solo, leading up to the grand finale, she gave a last supreme blast. As she did so, she fell from her chair with a cry and experienced a generalized convulsion. The hyperventilation in a potential epileptic had done its work. To date, seven years later, she has had no further trouble.

Recent work in electro-encephalography tends to show that this convulsive potentiality is present in certain individuals, and indications are that this type of work will be of great help in the understanding and prevention of these convulsive disorders.

The most noteworthy advance in recent medical therapy is the discovery of dilantin sodium which, unlike most drugs, does not produce drowsiness, but at the same time is most effective in reducing the number of seizures.[11]

SUMMARY

In this survey of the convulsive states, the following points have been emphasized:

1. The economic and social importance of caring for 600,000 epileptics.

2. The similarity in the fundamental mechanism of the convulsive state with sleep, faints and syncopy, as evidenced by carotid pressure, cortical stimulation, and clinical manifestations.

3. The momentous aid given by modern research to the understanding of these convulsive disorders.

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THE INTERPRETATION OF LABORATORY EXAMINATIONS IN THE DIAGNOSIS OF INFECTIOUS DISEASES*

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PART II†

ANOTHER type of infection that may be extremely acute and accompanied by vegetative endocarditis, which may give no murmurs, is that resulting from hemolytic streptococcal infection. The following case is an example.

A man, 44 years of age, develops fever, abdominal pain, and diarrhea, followed by jaundice, hematuria, nitrogen retention, and Streptococcus hemolyticus bacteremia. Death in ten days with acute bacterial endocarditis and focal embolic nephritis.

CASE 4.—This patient was in excellent health until four days before admission to the hospital. At that time he developed nausea and vomiting and shortly thereafter had abdominal cramps. The following day he had frequent loose bowel movements. The vomiting, abdominal cramps and diarrhea increased in severity until the time of admission. He also complained of chilliness and sweats, which appeared off and on for two days before admission.

Examination showed a well-developed individual who appeared acutely ill. He was mentally clear and coöperative. His temperature was 102 degrees Fahrenheit, pulse rate 110, blood pressure 120/60. Examination of the head, nose and throat proved negative. The heart and lungs were clear throughout. There were no cardiac murmurs. The abdomen showed slight tenderness over the upper half, but no masses were felt and the spleen was not palpable. The extremities showed an amputation stump of the left leg below the knee. Rectal examination was negative.

Urine examination showed no albumin, one or two white blood cells, and rare erythrocytes. The red blood cell count was 4,500,000, hemoglobin 81 per cent, white blood cell count 14,300. Nonprotein nitrogen was 50 milligrams per 100 cubic centimeters.

The clinical course in the hospital was one of progressive failure, the temperature varying between normal and 100 degrees Fahrenheit. On the fourth day after admission to the hospital, the seventh day of his illness, he became deeply jaundiced and the icteric index was recorded as 100 units. At the same time there was a progressive increase in his nitrogen retention. The urine contained no albumin, only a few white blood cells and red blood cells. On the fifth day the jaundice was increasing and the patient developed gross hematuria. The liver edge was felt two fingerbreadths below the costal margin; it was smooth but not especially tender. No murmurs appeared over the heart. Blood cultures, taken on the seventh, eighth and ninth days, showed hemolytic streptococci.

The clinical course, then, was that of a man who had been perfectly well until nine days before his death, when he was seized with abdominal pain, nausea and vomiting, and diarrhea. This was followed by fever, the development of jaundice, and the appearance of hematuria, with hemolytic streptococci in the circulating blood. Necropsy showed acute vegetative endocarditis of the aortic, mitral, and pulmonic valves and acute bronchopneumonia. In brief, this was an instance of hemolytic streptococcal sepsis with jaundice and vegetative endocarditis, focal embolic nephritis, and renal insufficiency. The nonprotein nitrogen of the blood increased from 50 to 188 milligrams per 100 cubic centimeters between the first and the seventh day of his illness. Here again is an instance of infection with bacteremia in which it was not possible to discover the primary focus or portal of entry. The course was extremely acute and produced lesions on the heart valves, which had not progressed to the stage where the leaflets were destroyed but were large enough to give origin to numerous emboli which went to the kidneys and produced a focal glomerular nephritis. The diagnosis of streptococcal infection during life would not have been made without blood culture.

* * *

The above cases illustrate how bacteremia aids one in making an etiologic diagnosis of an otherwise obscure infection. Blood cultures may also provide one with information when there is a mixed infection, such as is illustrated by the following case.

A common problem in diagnosis is the explanation of fever which recurs following a pneumococcal lobar pneumonia. While the possibilities must include the search for focal infections, i. e., empyema, endocarditis, or meningitis, very often there is an infection due to another organism, such as a hemolytic streptococcus, and the diagnosis is made by culturing the blood and examining the pleural fluid or sputum. Such cases, of which the following is an example, have been reported by Parsons and Myers,[10] Finland,[11] and Curphey and Solomon.[12]

A young man with pneumococcus Type I pneumonia and bacteremia fails to respond to large amounts of specific serum. Sputum contains large

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